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NEWS 3 AUG 18 COMPENDEX indexing changed for the Corporate Source (CS) field  
NEWS 4 AUG 24 ENCOMPLIT/ENCOMPLIT2 reloaded and enhanced  
NEWS 5 AUG 24 CA/CAplus enhanced with legal status information for U.S. patents  
NEWS 6 SEP 09 50 Millionth Unique Chemical Substance Recorded in CAS REGISTRY  
NEWS 7 SEP 11 WPIDS, WPINDEX, and WPIX now include Japanese FTERM thesaurus  
NEWS 8 OCT 21 Derwent World Patents Index Coverage of Indian and Taiwanese Content Expanded  
NEWS 9 OCT 21 Derwent World Patents Index enhanced with human translated claims for Chinese Applications and Utility Models  
NEWS 10 OCT 27 Free display of legal status information in CA/CAplus, USPATFULL, and USPAT2 in the month of November.

NEWS EXPRESS MAY 26 09 CURRENT WINDOWS VERSION IS V8.4,  
AND CURRENT DISCOVER FILE IS DATED 06 APRIL 2009.

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FILE 'MEDLINE' ENTERED AT 11:22:01 ON 09 NOV 2009

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=> s insulin(w)receptor(w)substrate(w)2 or IRS(w)2 and (activator or inhibitor)  
L1            3541 INSULIN(W) RECEPTOR(W) SUBSTRATE(W) 2 OR IRS(W) 2 AND (ACTIVATOR  
OR INHIBITOR)

=> s l1 and (over(w)express or over(w)produce)  
L2            2 L1 AND (OVER(W) EXPRESS OR OVER(W) PRODUCE)

=> dup rem l2  
PROCESSING COMPLETED FOR L2  
L3            2 DUP REM L2 (0 DUPLICATES REMOVED)

=> dis ibib abs 13

L3 ANSWER 1 OF 2 EMBASE COPYRIGHT (c) 2009 Elsevier B.V. All rights  
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ACCESSION NUMBER: 2005199769 EMBASE  
TITLE: The GLUTs family - Lessons from transgenic mice.  
AUTHOR: Hartil, K.; Weldon, R.H.; Seki, Y.; Charron, M.J.  
(correspondence)  
CORPORATE SOURCE: Department of Biochemistry, Albert Einstein College of  
Medicine, 1300 Morris Park Avenue, Bronx, NY 10461, United  
States. charron@aecon.yu.edu  
SOURCE: Current Medicinal Chemistry: Immunology, Endocrine and  
Metabolic Agents, (Apr 2005) Vol. 5, No. 2, pp. 189-206.  
Refs: 144  
ISSN: 1568-0134 CODEN: CMCIC8  
COUNTRY: Netherlands  
DOCUMENT TYPE: Journal; General Review; (Review)  
FILE SEGMENT: 016 Cancer  
              018 Cardiovascular Diseases and Cardiovascular Surgery  
              022 Human Genetics  
              029 Clinical and Experimental Biochemistry  
              003 Endocrinology  
              005 General Pathology and Pathological Anatomy  
LANGUAGE: English  
SUMMARY LANGUAGE: English  
ENTRY DATE: Entered STN: 19 May 2005  
              Last Updated on STN: 19 May 2005  
AB The glucose transporters (GLUTs) are currently a 13 member family of  
facilitative transmembrane proteins which transport glucose down its  
concentration gradient. The GLUTs have a tissue specific expression and  
regulation. Dysregulation of GLUTs have been implicated in the  
pathogenesis of a number of diseases including diabetes and cancer and are  
known to play an important role in the developing embryo. In addition,  
roles for GLUTs in cardiac function and embryonic development have been  
identified and will be discussed in this review. The ability to ablate or  
over-express GLUTs has advanced our understanding of the  
role these transporters play in the maintenance of normal glucose  
homeostasis and the pathogenesis of diabetes. The development of Cre-LoxP  
technology coupled with the existence of tissue specific promoters allows

investigators to manipulate gene expression both globally and in a tissue specific manner. The major GLUTs which have been investigated using transgenic technology are GLUT1, GLUT4 and GLUT2. Overexpression of GLUT4 and GLUT1 results in increased glucose uptake and metabolism. However, only GLUT4 overexpression protects against the development of insulin resistance in transgenic mice. Genetic ablation of GLUT4 and GLUT2 results in impaired insulin tolerance and defects in both lipid and glucose metabolism. This review will present various transgenic models of GLUT modification and discuss what has been learned from these models about the role that GLUTs play in glucose homeostasis, insulin action and development. .COPYRGT. 2005 Bentham Science Publishers Ltd.

=> dis ibib abs 13 2

L3 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2009 ACS on STN  
ACCESSION NUMBER: 2001:129407 CAPLUS  
DOCUMENT NUMBER: 134:261420  
TITLE: Specific inhibition by hGRB10 $\zeta$  of insulin-induced glycogen synthase activation: evidence for a novel signaling pathway  
AUTHOR(S): Mounier, C.; Lavoie, L.; Dumas, V.; Mohammad-Ali, K.; Wu, J.; Nantel, A.; Bergeron, J. J. M.; Thomas, D. Y.; Posner, B. I.  
CORPORATE SOURCE: The Polypeptide Hormone Laboratory, McGill University, Montreal, QC, H3A 2B2, Can.  
SOURCE: Molecular and Cellular Endocrinology (2001), 173(1-2), 15-27  
CODEN: MCEND6; ISSN: 0303-7207  
PUBLISHER: Elsevier Science Ireland Ltd.  
DOCUMENT TYPE: Journal  
LANGUAGE: English  
AB Grb10 is a member of a family of adapter proteins that binds to tyrosine-phosphorylated receptors including the insulin receptor kinase (IRK). In this study recombinant adenovirus was used to over-express hGrb10 $\zeta$ , a new Grb10 isoform, in primary rat hepatocytes and the consequences for insulin signaling were evaluated. Over-expression of hGrb10 $\zeta$  resulted in 50% inhibition of insulin-stimulated IRK autophosphorylation and activation. Anal. of downstream events showed that hGrb10 $\zeta$  over-expression specifically inhibits insulin-stimulated glycogen synthase (GS) activity and glycogen synthesis without affecting insulin-induced IRS1/2 phosphorylation, PI3-kinase activation, insulin like growth factor binding protein-1 (IGFBP-1) mRNA expression, and ERK1/2 MAP kinase activity. The classical pathway from PI3-kinase through Akt-PKB/GSK-3 leading to GS activation by insulin was also not affected by hGrb10 $\zeta$  over-expression. These results indicate that hGrb10 $\zeta$  inhibits a novel and presently unidentified insulin signaling pathway leading to GS activation in liver.  
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REFERENCE COUNT: 47 THERE ARE 47 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

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L1 3541 SEA FILE=MFE SPE=ON ABB=ON PLU=ON INSULIN(W) RECEPTOR(W)

SUBSTRATE(W) 2 OR IRS(W) 2 AND (ACTIVATOR OR INHIBITOR)  
L2 2 SEA FILE=MFE SPE=ON ABB=ON PLU=ON L1 AND (OVER(W) EXPRESS  
OR OVER(W) PRODUCE)  
L3 2 DUP REM L2 (0 DUPLICATES REMOVED)  
DIS IBIB ABS L3  
DIS IBIB ABS L3 2  
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